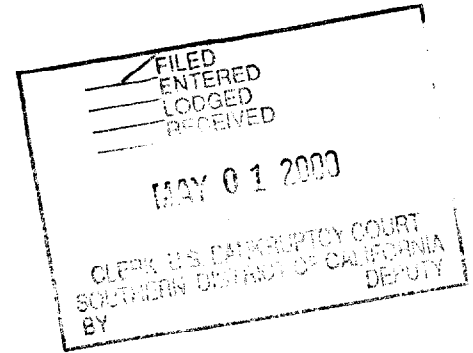


1 L. Scott Keehn (SBN 61691)  
2 Lisa L. Keehn (SBN 167696)  
3 **ROBBINS & KEEHN**  
4 A Professional Corporation  
5 530 "B" Street, Suite 2400  
6 San Diego, California 92101  
7 Telephone: (619) 232-1700

8 Attorneys for Debtor  
9 **SARA NEWSOME BURNS**



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**UNITED STATES BANKRUPTCY COURT**  
**SOUTHERN DISTRICT OF CALIFORNIA**

In re:  
  
SARA NEWSOME BURNS, an individual,  
  
Debtor.

CASE NO. 99-33191-B7

DECLARATION OF L. SCOTT KEEHN  
IN OPPOSITION TO MOTION  
OBJECTING TO DEBTOR'S  
AMENDED EXEMPTION CLAIM

Date: May 30, 2000  
Time: 11:00a.m.  
Dept: 4  
Hon. Peter W. Bowie

I, L. SCOTT KEEHN declare as follows:

1. I am an attorney at law licensed to practice law in the State of California, and admitted to practice before the United States District Court for the Southern District of California. I am also a shareholder in the firm of Robbins & Keehn, a Professional Corporation, attorneys of record for Sara N. Burns, the Debtor in the above entitled bankruptcy proceedings. I am the shareholder in charge of this engagement, and the attorney within the Firm most familiar with the engagement and matters related to it.

2. Attached hereto marked Exhibit 1 and incorporated herein by this reference are the title/copyright notice pages of *Mosby's Handbook of Diseases*, Mosby-Yearbook, Inc. (1996), and pages 252-255 of that handbook which relates to Hepatitis.

ROBBINS & KEEHN, APC  
ATTORNEYS AT LAW  
2400 UNION BANK BUILDING - 530 "B" STREET  
SAN DIEGO, CALIFORNIA 92101  
TELEPHONE (619) 232-1700 - TELECOPIER (619) 544-9095

ORIGINAL

4. I have first hand knowledge of all of the foregoing, and if called as a witness could, and would, testify in the manner herein above set forth.

L. Scott Keehn  
L. SCOTT KEEHN

# *Mosby's* Handbook of Diseases

**RAE LANGFORD, EdD, RN**  
Private Practice,  
Rehabilitation Nurse Consultant,  
Houston, Texas

**JUNE M. THOMPSON, DrPH, RN**  
New Mexico Department of Health,  
Division of Epidemiology,  
Santa Fe, New Mexico;  
Clinical Nursing Affiliations,  
University of New Mexico  
Health Sciences Center Hospital,  
Albuquerque, New Mexico

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 Senior Editor: Sally Schreier  
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 Project Manager: Patricia Tannian  
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## Consultants

Carolyn Cohen Parchinsky, MA, RN, CS  
 Instructor, School of Nursing,  
 Holy Name Hospital,  
 Teaneck, New Jersey

Carol Ruscini, MSN, RNP  
 Case Manager/Geriatric Clinical Nurse Specialist,  
 Department of Health Affairs,  
 Health Advantage Hospital,  
 Little Rock, Arkansas

Jeannette L. Sasmor, ASS, MED, EdD, MBA  
 MCH Coordinator,  
 Department of Nursing & Allied Health,  
 Yavapa College,  
 Prescott, Arizona

Patricia R. Teasley, MSN, RN, CS  
 Associate Professor, Department of Nursing  
 Columbus College,  
 Columbus, Georgia

**EXHIBIT 1**

oping countries in Africa, Asia, or Central America. It is most severe in pregnant women.

**Pathophysiology:** The etiologic agent, mode of transmission, and clinical course vary according to the hepatitis type. However, the pathophysiology is the same. The causative agent invades the mononuclear cells in the liver, replicates, and sets up an inflammatory process in the parenchyma and portal ducts, causing hepatic cell necrosis, cellular collapse, and accumulation of necrotic tissue in the lobules and portal ducts. This results in interference with bilirubin excretion. Cellular regeneration and mitosis occur simultaneously with cellular necrosis, and the liver regenerates within 2 to 3 months. Continuation of the inflammatory response sets up a chronic disease process.

EXHIBIT 1

## Hepatitis (Viral)

—A diffuse inflammation of the cells of the liver that produces liver enlargement and jaundice.

**Etiology and Incidence:** The cause is a variety of hepatotropic viruses. To date, five viral types that cause primary hepatitis have been positively identified; these viruses are known as hepatitis A (HAV), hepatitis B (HBV), hepatitis C (HCV), hepatitis D (HDV), and hepatitis E (HEV). Viruses F and G have been discovered and may also cause primary hepatitis. Other viruses tentatively labeled as GB-A, GB-B, and GB-C are being tested to see if they differ from F and G and if they also cause hepatitis. HAV is transmitted by contaminated food and water and by the fecal-oral route; HBV and HDV are transmitted by contact with bodily fluids, and HCV by percutaneous exposure to blood, and HEV by contaminated water and the fecal-oral route. **Note:** Hepatitis may also occur as a secondary infection and is associated with viruses from other primary diseases, including cytomegalovirus, Epstein-Barr, herpes simplex, varicella-zoster, coxsackie B, and rubella viruses.

Primary viral hepatitis occurs worldwide. More than 70,000 cases are reported annually in the United States, and the incidence is rising. Hepatitis A is seen most often in children and young adults, but the incidence is rising in those who are HIV positive. Hepatitis B affects all age groups; about 10% of all transfusion-related hepatitis is this type. Hepatitis C accounts for about 20% of all cases and for most transfusion-related cases. It is seen across all age groups. Hepatitis D is seen in individuals who are susceptible to HBV or may be HBV carriers, such as hemophiliacs and IV drug users. The disease manifestation is severe in children. Hepatitis E is seen primarily among young adults in devel-

## 254 Hepatitis

## Clinical Manifestations

Incubation	HAV: 15 to 50 days; HBV: 45 to 180 days; HCV: 14 to 182 days; HDV: 14 to 70 days; HEV: 15 to 64 days
Communicability	HAV: last half of incubation until 1 week after onset of jaundice; HBV: during incubation and entire clinical course (carrier state may persist for years); HCV: 1 week before clinical onset to indefinite period of time as carrier; HDV: throughout clinical disease; HEV: unknown
Preicteric phase	Malaise, headache, nausea and vomiting, anorexia, myalgia, chills, fever, upper quadrant abdominal pain; HBV, HDV: hives, itching, erythema, arthritis
Icteric phase	Appetite returns; malaise continues; jaundice with or without itching; dark urine; clay-colored stools

**Complications:** Complications include development of chronic hepatitis, spontaneous relapse, and cirrhosis. Severe fulminant hepatitis with rapid cellular destruction, no regeneration, and accompanying encephalopathy occur in 1% of cases and are usually fatal.

**Diagnostic Tests:** Serum enzymes (aspartate aminotransferase [serum glutamic oxaloacetic transaminase], alanine aminotransferase [serum glutamic pyruvic transaminase]) 8 to 20 times normal values during the prodromal and clinical

## Hepatitis 255

phases, lactate dehydrogenase is 1 to 3 times normal. These elevations are the hallmark of the disease. The differential diagnosis is based on the clinical history and various laboratory tests. In HAV, the stool is positive for the virus 2 to 4 weeks after exposure, and the enzyme-linked immunosorbent assay (ELISA) shows a rise in HAV antibodies. In HBV, serum antigen tests detect HBsAg, and serum antibody tests detect a rise in anti-HBc. A serum test for HCV is now available.

## Therapeutic Management

Surgery	None
Drugs	Immune globulin for prophylaxis in those exposed to HAV and HBV; vaccine for prophylaxis in individuals exposed to or at high risk of contracting HBV; antiemetics (chlorpromazine is contraindicated in hepatic disease) for nausea; analgesics for pain (acetaminophen is preferred)
General	Bed rest; diet as tolerated, with frequent, small, low-fat, high-carbohydrate meals; adequate fluid intake; appropriate infection precautions, dictated by transmission routes; monitoring by liver function tests until normal value is achieved

EXHIBIT

1

Source

## Hepatitis C

Prepared by  
Judy D. Gassett, MPH, CIC  
Infection Control Advisor

## Viral Hepatitis

Prepared by  
Judy D. Gassett, MPH, CIC  
Infection Control Advisor

- Hepatitis A 1973
- Hepatitis B 1970
- Hepatitis C 1989
- Hepatitis D 1977
- Hepatitis E 1980
- Hepatitis F 1994
- Hepatitis G 1995

## History of Hepatitis C

- In 1989 the CDC identified transfusion related hepatitis, (the hepatitis often seen after a blood transfusion), as being hepatitis C. Prior to 1989, it was called "Non A, Non B" hepatitis.

## Hepatitis C: Epidemiology

- 4 million Americans or 2% now positive for HCV!
- About 9,000 deaths yearly.
- In the US 75% of patients have the type 1 virus which is most often associated with chronicity and refraction to treatment/cure.
- In the 1980's 180,000 annual cases/yr. In the 1990's around 35,000 new cases/yr because new lab tests made protection of the blood supply possible.

### Hepatitis C: Epidemiology

- Of 100 pts who are infected with the hep C virus.
  - 15 recover (but are NOT IMMUNE!).
  - 1 will develop fulminant hepatitis with / wo death.
  - 84 develop persistent viremia (remain infected & have chronic disease).
    - 30 will become chronically infected and develop symptoms including portal hypertension, cirrhosis, & hepatocellular carcinoma.

### Hepatitis C: Epidemiology

- Of the 84 developing persistent viremia (remain infected & have chronic disease).
  - These 30 with symptoms are the ones that usually end up needing a liver transplant. Hepatitis C is the leading reason for liver transplants.
  - 54 don't know that they are infected become chronic carriers (can transmit it to others). Disease usually does not progress quickly.

### Risks of Transfusion Associated Non-a Non-b Hepatitis (Hepatitis C)

- Risk of getting hep C from donor blood prior to 1992 was 5% or 1 in 20%! A huge and unacceptable risk!
  - One in 200 units was infected.
- 1986-1990 "surrogate markers" (elevated liver enzymes) were used to screen donor blood. Units with elevated enzymes were rejected.
  - Transmission from blood remained high because liver enzymes are normal 30% of the time in hep C. We needed a good blood screening test.

### How the American Blood Supply Got Cleaned Up From Hep C

- In 1991-92 was the first wide-scale testing for presence of hepatitis C antibody in donor blood.
  - Dramatically reduced hep C risk from transfused blood to less than one in 1,000 units.
  - Since 1002, with better tests blood supply even safer now! Less than 1 in 50,000 units infected with hep C.

### Hepatitis C Molecular Biology Problems and Implications

- Virus has poor RNA reading ability. When it replicates, at least one mutation usually in the viral envelope occurs resulting new viral species! (HIV does this also).
- Over time the patient has hundreds of "quasi species".
- Quasi species can escape immunological surveillance.
- Vaccines are impossible to develop against all the variations.
- Infestation of lymphoid tissue allows virus to persist for as long as the host lives.

### Implications Of the Biology Of The Hepatitis C Virus

- Antibodies are markers for the virus; they are NOT protective. Anyone with hepatitis C antibodies should be considered infected.
- You can get hepatitis C again after having recovered. Current molecular methods probably cannot detect that this has occurred. But if you cannot develop protective antibody then you can be re-infected.
- We are not sure that persons who recover from hep C (and are not carriers), will not revert to the carrier state some point in the future.

### Biggest Risk Groups

- 80% of drug abusers are infected with hepatitis C1
  - IV drug users.
  - Nasal cocaine abusers (nasal straw is shared).
- Persons who have received blood components, especially before 1992. Hepatitis C is most often found in:
  - 60% of thalassemia (severe anemia).
  - 90% hemophilia.
  - 15% of hemodialysis.

### Transmission Precautions for Hepatitis C

- National Institutes of Health (NIH) does not recommend condoms to avoid transmission.
- Vertical transmission thought to be low risk. Infected mothers can nurse. (Transmission will probably be found to be related to Mother's viral load).
- You can share plates and utensils, combs, brushes but not toothbrushes and razors, which touch body substances. In BSI, making this distinction does not matter since you always avoid contact with another person's body substances.

### What To Do For Needle Stick Injuries

- Obtain baselines for hepatitis C antibody and liver enzymes (ALT), and again at 6 months. If positive confirm with more advanced test.
- Get permission to test the source patient for hep C antibody. If positive, consider a viral load study on the source.
- Interferon does not work for prophylaxis and immune globulin has never been recommended for hep C.

13.

### What To Do For Needle Stick Injuries

- It is important to document testing for hepatitis C when HCW are injured. HCW need to document acquisition was work related to insure that he or she will have costly treatment provided at no cost.
- Wash site of exposure with soap and water; bleaching, betadine, lancing and pumping blood out of your finger is worthless and painful.
- Interferon/ribavirin may have a role in curing or making the disease less severe, but has no role in prophylaxis (preventing disease).

14.

### Rate of Progression of Hepatitis C to Cirrhosis and Cancer

- All persons with hep C eventually develop cirrhosis if they live long enough.
- Certain factors increase progression to cirrhosis/liver cancer include:
  - Alcohol consumption in a hep C positive person.
  - Co-infection with another hepatitis virus.
  - Development of an unrelated disorder than stresses immune defenses (diabetes, leukemia, etc).

15.

### Tools For Evaluating Hepatitis C

- ALT levels are useless for determining the severity of liver disease in pts with hep C.
- Liver enzymes are often normal in patients who have active hep C & cirrhosis. Liver function tests (bilirubin, protime, albumin) are also often normal even with advanced cirrhosis.
- The liver biopsy correlates most closely with how hep C is progressing, disease severity, and how the pt is doing clinically! It is the only method of evaluating disease severity and activity.

16.

### Who Should Be Treated for Hep C? Current Clinical Thinking

- Since Hep C is slowly progressive for most persons, not everyone should be treated (tx is expensive, and the drugs have side effects). The current thinking is to get a liver biopsy.
  - Biopsy = inflammation and fibrosis: Treat (pt is progressing to cirrhosis and its complications).
  - Biopsy = normal histology: Don't treat (disease not progressing); but watch patient.
  - If liver enzymes high but biopsy OK: Don't treat; watch patient.

### Drugs To Treat Hepatitis C

- Initial response to interferon is < 40%.
- When tx stopped 75% of pts relapse!
- Adding ribavirin (non specific anti-viral for RNA viruses) improves response rate to over 50%.
  - (Ribavirin always causes hemolysis and patients will become anemic taking it.
- The longer the person has hep C, the less likely to respond to tx.
- If patient has hepatitis C, immunize for hep B and A!

### Combo Therapy (Interferon + Ribavirin) For Hepatitis C

- Ribavirin PREVENTS relapse (defined as absence of HCV virus in the blood) when used with interferon.
- Ribavirin + interferon = response in > 50% of patients and the virus stays away for > year. (No published studies beyond a year yet).
- Interferon alone = long term response <20% of pts. For most patients, as soon as interferon stopped, virus comes right back.

### Combo Therapy (Interferon + Ribavirin) For Hepatitis C

- Studies using ribavirin were done in Europe where the viral types 2 and 3 are prevalent and easier to treat.
  - Persons with low viral load also had better response rate.
- In US, (where type 1 virus is prevalent), we may not get as high a response rate but it will be better than interferon alone!

### Hepatitis F

- Hepatitis F virus does not exist.
- In 1994 there was a brief report of a new enteric virus reported in the *Journal of Virology*.
- It turns out that this "virus" could not be re-isolated and is not a new virus.
- Therefore, we have an opening for hepatitis F.

21

### Hepatitis G

- This is a virus in search of a disease.
- Dr Harvey Alter studied it in 1997 and found antibody to G in most patients with post transfusion hepatitis C.
- It seems to be transmitted via blood products and leads to persistent viremia.
- It does not increase the symptoms or consequences of having hepatitis C, as far as we can tell.

22

### Hepatitis G

- In the last 3 years at the "liver meetings", many papers have shown that persons with hep G are NOT more likely to develop cirrhosis or fulminant hepatitis than with hep virus C alone.
- At this time there is no proof that hep G replicates in the liver or causes any disease. The question proposed by Dr. Alter is whether or not hepatitis G is a virus or a phenomenon.
- We may also loose hepatitis G.

23

Springhouse Corp.  
1985

Nurse's Reference Library: Diseases

## 730 HEPATOBILIARY DISORDERS

dration without inducing vomiting.

- Record weight daily, and keep accurate intake and output records. Observe feces for color, consistency, frequency, and amount.
- Watch for signs of hepatic coma, dehydration, pneumonia, vascular problems, and decubitus ulcers.
- In the patient with fulminant hepatitis, maintain electrolyte balance and a patent airway, control bleeding, and correct hypoglycemia and other compli-

cations while awaiting liver regeneration and repair.

- Explain the nature of hepatitis so the patient understands the need for isolation. Explain all diagnostic tests.
- Before discharge, urge the patient to have regular medical checkups for at least 1 year. Warn him not to drink any alcohol during this time, and tell him how to recognize recurrence. Refer the patient for follow-up, as needed.

—DEBORAH STEINER, RN

## Nonviral Hepatitis

*Nonviral inflammation of the liver (toxic or drug-induced hepatitis) is a form of hepatitis that usually results from exposure to certain chemicals or drugs. Most patients recover from this illness, although a few develop fulminating hepatitis or cirrhosis.*

### Causes

Various hepatotoxins—carbon tetrachloride, trichloroethylene, poisonous mushrooms, vinyl chloride—can cause the toxic form of this disease. Following exposure to these agents, liver damage (diffuse fatty infiltration of liver cells and necrosis) usually occurs within 24 to 48 hours, depending on the size of the dose. Alcohol, anoxia, and preexisting liver disease exacerbate the toxic effects of some of these agents.

Drug-induced (idiosyncratic) hepatitis may stem from a hypersensitivity reaction unique to the affected individual, unlike toxic hepatitis, which appears to affect all persons indiscriminately. Among the drugs that may cause this type of hepatitis are halothane, sulfonamides, isoniazid, alpramethyldopa, and chlorpromazine (cholestasis-induced hepatitis). In hypersensitive persons, symptoms of hepatic dysfunction may appear at any time during or after exposure to these drugs but usually emerge after 2 to 5 weeks of therapy. Not all adverse drug reactions are toxic. Oral contraceptives, for example, may impair liver function and produce jaundice without causing necrosis, fatty infiltration of liver cells,

or a hypersensitive reaction.

### Signs and symptoms

Clinical features of toxic and drug-induced hepatitis vary with the severity of liver damage and the causative agent. In most patients, symptoms resemble those of viral hepatitis: anorexia, nausea, vomiting, jaundice, dark urine, hepatomegaly, possible abdominal pain (with acute onset and massive necrosis), and clay-colored stools or pruritus with the cholestatic form of hepatitis. In addition, carbon tetrachloride poisoning produces headache, dizziness, drowsiness, and vasomotor collapse; in halothane-related hepatitis, fever, moderate leukocytosis, and eosinophilia; with chlorpromazine, abrupt fever, rash, arthralgias, lymphadenopathy, and epigastric or upper right quadrant pain.

### Diagnosis

Diagnostic findings include elevations in serum transaminase (SGOT, SGPT), both total and direct bilirubin (with cholestasis), alkaline phosphatase (unusually high), WBC, and eosinophils (possible in drug-induced type). Liver biopsy may help identify the underlying pathology,

## LIVER DISEASES 731

especially infiltration with WBCs and eosinophils. Liver function tests have limited value in distinguishing between nonviral and viral hepatitis.

#### Treatment and nursing intervention

Effective treatment must remove the causative agent by lavage, catharsis, or hyperventilation, depending on the route of exposure. Dimercaprol may serve as an antidote for toxic hepatitis caused by

gold or arsenic poisoning but doesn't prevent drug-induced hepatitis caused by these substances. Corticosteroids may be ordered for patients with the drug-induced type. Thiocetic acid, an investigational drug, has been tried successfully for mushroom poisoning. Preventive nursing measures should include instructing the patient about the proper use of prescription drugs, and the proper handling of cleaning agents and solvents.

—JUANITA WATSON, RN, MSN

## Cirrhosis and Fibrosis

*Cirrhosis is a chronic hepatic disease characterized by diffuse destruction and fibrotic regeneration of hepatic cells. As necrotic tissue yields to fibrosis, this disease alters liver structure and normal vasculature, impairs blood and lymph flow, and ultimately causes hepatic insufficiency. It's twice as common in men as in women and is especially prevalent among malnourished chronic alcoholics over age 50. Mortality is high; many patients die within 5 years of onset. Prognosis is better in noncirrhotic forms of hepatic fibrosis, which cause minimal hepatic dysfunction and don't destroy liver cells.*

#### Causes

The following clinical types of cirrhosis reflect its diverse etiology:

- *Portal, nutritional, or alcoholic cirrhosis* (Laennec's type), the most common, accounts for 30% to 50% of patients, up to 90% of whom have a history of alcoholism. Liver damage results primarily from malnutrition, especially of dietary protein, which causes scar tissue to form around the portal area.
- *Biliary cirrhosis* (15% to 20% of patients) results from bile duct diseases, which suppress bile flow.
- *Postnecrotic (posthepatic) cirrhosis* (10% to 30% of patients) stems from various types of hepatitis.
- *Pigment cirrhosis* (5% to 10% of patients) may stem from disorders such as hemochromatosis.
- *Cardiac cirrhosis* (rare) refers to liver damage caused by right heart failure.
- *Idiopathic cirrhosis* (about 10% of patients) has no known cause.

Noncirrhotic fibrosis may result from schistosomiasis or congenital hepatic fi-

brosis, or may be idiopathic.

#### Signs and symptoms

Clinical manifestations of cirrhosis and fibrosis are similar for all types, regardless of cause. Early indications are vague but usually include gastrointestinal symptoms (anorexia, indigestion, nausea, vomiting, constipation, or diarrhea) and dull abdominal ache. Major and late symptoms develop as a result of hepatic insufficiency and portal hypertension, and involve the entire body. These clinical features include:

- *respiratory*—limited thoracic expansion due to abdominal ascites, interfering with efficient gas exchange and leading to hypoxia
- *CNS*—various symptoms of hepatic encephalopathy, including lethargy, mental changes, slurred speech, asterixis, peripheral neuritis, paranoia, and hallucinations, progressing to extreme obtundation and coma
- *hematologic*—bleeding tendencies (nosebleeds, easy bruising, bleeding

## LIVER DISEASES

### Viral Hepatitis

A fairly common systemic disease, viral hepatitis is marked by liver cell destruction, necrosis, and autolysis, leading to anorexia, jaundice, and hepatomegaly. More than 70,000 cases are reported annually in the United States. This disease has three forms: type A (infectious or short-incubation hepatitis), type B (serum or long-incubation hepatitis), and type non-A, non-B hepatitis. All three types are found worldwide. Type B hepatitis rarely occurs in epidemics but has a higher mortality than type A hepatitis, which tends to be benign and self-limiting. Type non-A, non-B hepatitis has the mildest course.

#### Causes and incidence

Type A hepatitis is highly contagious and is usually transmitted by the fecal-oral route, although occasionally it's transmitted parenterally. The most common cause is ingestion of contaminated food, water, or milk. Outbreaks of type A hepatitis often occur after people have eaten seafood that came from polluted water. Type B hepatitis, which is generally transmitted parenterally, can also be spread through contact with human secretions and feces. Nurses, doctors, laboratory technicians, blood bank workers, and dentists are frequent victims of type B hepatitis, often as a result

of wearing defective gloves while working. In addition, the incidence of both type A and type B hepatitis appears to be rising among homosexuals, presumably because of oral and anal sexual contact. Type non-A, non-B hepatitis accounts for 60% to 90% of post-transfusion hepatitis in the United States, and transmission usually results from commercial blood donations.

In most patients with hepatitis, liver cells eventually regenerate with little or no residual damage. Patients usually recover readily, with a lifelong immunity to type A hepatitis (but not to type B). Old age and serious underlying disor-

#### DIFFERENCES BETWEEN HEPATITIS A AND B

	TYPE A (infectious)	TYPE B (serum)
Age incidence	Children, young adults	Any age
Seasonal incidence	Fall, winter	Any time
Transmission	Food, water, semen, tears, stools, and possibly urine	Serum, blood and blood products, and semen
Incubation	15 to 45 days	40 to 180 days
Onset	Sudden	Insidious
Serum markers	Anti-HAV	HB Ag + and HB
Prognosis	Good	Worsens with age
Carrier state	No	Yes

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STEINER, RN

## 728 HEPATOBILIARY DISORDERS

ders (congestive heart failure, severe anemia, diabetes, malignancy) make complications more likely. Prognosis is poor if edema and hepatic encephalopathy develop.

**Signs and symptoms**

The preicteric phase of viral hepatitis begins with a wide range of clinical features: fatigue, malaise, arthralgia, myalgia, headache, anorexia, photophobia, pharyngitis, cough, and coryza. This disease also causes nausea and vomiting, often with alterations in the senses of taste and smell; the patient may lose all desire to drink alcohol or smoke. Fever, with temperature of 100° to 101° F. (37.8° to 38.3° C.), may be associated with liver and lymph node enlargement. Symptoms begin suddenly in type A hepatitis and insidiously in type B; they disappear with the onset of jaundice. Type non-A, non-B hepatitis has a clinical course similar to type B hepatitis but is milder.

Mild weight loss, dark urine, clay-colored stools, and yellow scleras and skin signal the start of the icteric phase of hepatitis. In this second phase, anorexia may continue, the liver remains enlarged and tender, and the patient complains of discomfort and pain in the right upper abdominal quadrant. During this phase, splenomegaly, cervical adenopathy, and bile obstruction (cholestatic hepatitis) may develop; the patient may also be irritable and experience severe pruritus.

Jaundice, which may last from 1 to 2 weeks, results from the damaged liver cells' inability to remove bilirubin from the blood but doesn't indicate the severity of the disease. Occasionally, hepatitis occurs without jaundice (anicteric hepatitis). After jaundice disappears, the patient continues to experience fatigue, flatulence, abdominal pain or tenderness, and indigestion, although appetite usually returns and liver enlargement subsides. The posticteric, or convalescent phase generally lasts from 2 to 6 weeks, with full recovery in 6 months. Complications include chronic hepa-

titis, which may be benign (chronic persistent hepatitis) or active (chronic aggressive hepatitis). About 25% of patients with chronic aggressive hepatitis die from hepatic failure. Fulminant hepatitis, a life-threatening complication, develops in about 1% of patients, causing unremitting hepatic failure, with encephalopathy. Progression is commonly to coma and death within 2 weeks.

**Diagnosis**

Patient history revealing recent exposure to drugs, chemicals, or jaundiced persons, or recent blood transfusions or injections, in the presence of typical clinical features, strongly suggests viral hepatitis. Recent piercing of the patient's ears may also be significant, since contaminated instruments can cause hepatitis.



The presence of hepatitis B surface antigens (HBsAg) and hepatitis B antibodies (anti-HBs) confirms a diagnosis of type B hepatitis. HBsAg—sometimes called Australia antigen because it was originally discovered in the serum of an Australian aborigine—appears early in the disease, but blood levels may be negative later, giving a false negative reading if drawn too late. Detection of an antibody to type A hepatitis (anti-HAV) confirms diagnosis.

In the presence of HBsAg, anti-HBs, and anti-HAV, other laboratory results support a diagnosis of viral hepatitis, type A or type B; in their absence, these tests confirm type non-A, non-B hepatitis:

- prolonged prothrombin time (more than 3 seconds longer than the normal 12 to 15 seconds indicates severe liver damage)
- elevated SGOT and SGPT levels and slightly elevated serum alkaline phosphatase, reflecting the presence of enzymes in the blood
- elevated serum and urine bilirubin (with jaundice)
- low serum albumin and high serum globulin
- increased cephalin flocculation and thymol turbidity levels

- liver biopsy and scan showing patchy necrosis.

Hepatitis may be mistaken for infectious mononucleosis, although patients with mononucleosis have more prominent lymphadenopathy. During the preicteric phase of acute type B hepatitis, a serum sickness-like syndrome sometimes occurs, causing arthralgia, arthritis, rash, angioedema, and sometimes, hematuria and proteinuria, which may be misdiagnosed as rheumatoid arthritis or lupus erythematosus.

#### Treatment

No specific treatment exists for hepatitis. The patient should rest—at least in the early stages of the illness—and combat anorexia by eating small meals high in calories and protein. (Protein intake should be reduced if signs of precoma—lethargy, confusion, mental changes—develop.) Large meals are usually better tolerated in the morning. Antiemetics (trimethoprim or benzquinamide) may be given ½ hour before meals to relieve nausea and prevent vomiting; phenothiazides have a cholestatic effect and should be avoided. If vomiting persists, the patient will require I.V. infusions.

In severe clinical hepatitis, administration of corticosteroids may give the patient a sense of well-being and stimulate appetite, while decreasing itching and inflammation. Corticosteroids should be given sparingly, however, since their use in hepatitis is controversial.

All cases of hepatitis should be reported to public health officials. Ask the patient to name anyone he contacted recently who might have contracted the disease.

#### Nursing intervention

Design your care plan around supportive care, close observation, and emotional support.

- Isolate the patient in a single room with a private bath. Wear gloves when handling fluids and feces, and when drawing blood from a patient with type B hepatitis. Line wastebaskets with dis-

#### PREVENTION OF VIRAL HEPATITIS

Immune serum globulin (ISG)—commonly known as gamma globulin—has been studied extensively and found to be 80% to 90% effective in preventing type A hepatitis when promptly and properly administered. In confirmed type A cases, ISG should be given as soon as possible after exposure but within 2 weeks after onset of jaundice. ISG should also be administered prophylactically to contacts who may have been exposed. Transmission is usually through the fecal-oral route, and persons at high risk include household, intimate, sexual, and institutional contacts. Natural disease is believed to produce permanent immunity.

Most gamma globulin manufactured in the U.S. contains low titers of antibody against type B hepatitis (anti-HBs), which probably transmits some passive protection against type B hepatitis. A high-titer hepatitis B immune globulin (HBIG) is also available and is approximately 70% effective in preventing type B hepatitis. A recently developed vaccine for type B hepatitis shows great promise. It's made from fragments of the virus' surface coat, which stimulate the body's defenses to produce immunity. High-risk factors include oral or percutaneous contact with an HBsAg-positive fluid or sexual contact within the 4 weeks before onset of jaundice.

—MARRYN R. SHANAN, RN, MS

posable plastic bags. See that the patient wears pajama bottoms, to minimize contamination of bedsheets.

- Limit visitors and impose isolation precautions.

- Encourage the patient to eat as much as possible. Don't overload his meal tray; too much food on the tray will only diminish his appetite. And don't overmedicate; this too will diminish his appetite. Force fluids (at least 4,000 ml/day). Encourage the anorectic patient to drink fruit juices. Also, offer chipped ice and effervescent soft drinks to maintain hy-

## Jaundice

Jaundice is caused by abnormal accumulation of the pigment bilirubin (see page 466) in the blood and body tissues. This yellow-brown substance, which is produced from breakdown of old blood cells, is metabolized by the liver and excreted into the bile to be passed out of the body via the intestinal tract. Bacterial action on bilirubin in the gut is what gives the stool its normal brown color. When bilirubin cannot be passed off by the normal means, it accumulates in the liver and tissues throughout the body. The stool may become very pale, almost colorless, and a yellowish cast develops in the skin and in the whites of the eyes. This is why jaundice used to be referred to as "yellow jaundice."

Jaundice commonly occurs when a blockage of the bile duct causes a backup of bile and thus of bilirubin. But there are other causes as well. A severely inflamed or damaged liver may not be able to process the bilirubin delivered to it. Or production of bilirubin by the rapid destruction of red blood cells may become excessive, and the capacity of the liver may be overwhelmed. In examining a jaundiced patient the doctor must decide whether the patient has excess bilirubin production (hemolysis of red blood cells), defective metabolism of the bilirubin within the liver (seen in hepatitis), or defective flow of bile because of a bile-duct blockage. It is sometimes easy to distinguish between these possibilities, but in about 15 to 20 percent of the cases it is quite difficult.

Doctors often classify jaundice into two types: surgical jaundice (caused by blockage of the bile ducts) or medical jaundice (hemolysis, hepatitis, etc.). Persons with surgical jaundice usually are relieved of their symptoms promptly by surgery, but those with medical jaundice will not benefit from (and may be harmed by) an operation. Recently it has become considerably easier to look at bile ducts to establish whether they are blocked without resorting to surgery. Endoscopy has provided us with a test called endoscopic retrograde cholangiopancreatography (ERCP) (see page 467). An endoscope is passed into the duodenum to the area where the bile ducts and pancreatic duct enter the duodenum. A small catheter or flexible tube is inserted into this opening (called the ampulla of Vater) and dye is injected into the ducts. When successful, this procedure clearly discloses the bile ducts and can show the nature of any obstruction. Ultrasound waves also can be used to examine

obstructed ducts, as can a CAT scanner (see chapter 32, "X Rays and Radiology"). Finally, the bile ducts may be examined by inserting a very thin needle through the skin into the liver until a bile duct is punctured, after which dye is injected. These tests ordinarily are not all ordered simultaneously for a single patient, but are options.

## Hepatitis

Hepatitis means inflammation of the liver tissue, which may be caused by toxins, drugs, radiation, or most commonly, viral infection (see chapter 28, "Infectious Diseases"). The illness may occur in epidemic form, and the culprit is usually the so-called hepatitis A virus. Victims are often extremely ill, but the disease rarely causes severe destruction of the liver, liver failure, or the degenerative changes of cirrhosis. It is rarely, if ever, fatal.

Spread by oral ingestion of the virus, hepatitis A is highly contagious. Because of its epidemic nature, it is often easy to diagnose. If a number of people develop hepatitis simultaneously, hepatitis A is usually suspected. The patients may complain of weakness, fatigue, dark urine, light stools, itching skin, and jaundice. Blood tests reveal a characteristic pattern of liver abnormalities. It is sometimes possible to test the blood for the presence of antibodies to hepatitis A. Treatment consists of "taking it easy," but bed rest is not necessary. A nutritious diet is recommended, but some patients are so nauseated that they can eat very little. Hospitalization is rarely necessary. Strict hygiene is warranted. The patient should use separate plates and utensils and a separate bathroom, if possible. However, the patient with hepatitis A is at his most infectious just *before* his illness becomes apparent. When the telltale signs of jaundice appear, the virus particles are no longer present in the patient's stool and he can no longer infect persons around him.

Hepatitis B is often indistinguishable from hepatitis A at first glance. The same symptoms and the same pattern of liver test abnormalities occur. There are, however, several specialized tests that can detect hepatitis B virus in the blood. The most common test is for the hepatitis B antigen (Australia antigen).

Antibodies developed by the patient after contact with the hepatitis B virus can also be determined.

The route of infection by hepatitis B is almost always different from that of hepatitis A. Hepatitis B is usually caused by transfusion (or other inoculation) with contaminated blood. There is also increasing evidence that the disease may be spread by sexual contacts. A mother with hepatitis B at the time of labor and delivery stands a chance of transmitting the virus to the baby. Other people at risk are illicit drug users who share needles used by infected people.

Treatment for hepatitis B is the same as for hepatitis A. The outcome is generally just as favorable, but an occasional patient will develop severe liver disease with liver failure. Further, patients who have hepatitis B may go on to develop a chronic form of hepatitis.

Not all cases of hepatitis transmitted by blood transfusions are caused by the hepatitis B virus. One or more additional viruses obviously exist. The disease caused by these other viruses is referred to as non-A, non-B hepatitis (or hepatitis C). The best designation, however, seems to be simply post-transfusion hepatitis. The disease produced by these organisms is quite similar to that produced by hepatitis B. Several other viruses are occasional causes of hepatitis. Active research is being carried on in this field.

**Chronic active hepatitis**, unlike acute hepatitis, implies a long-term illness characterized by continued inflammation and gradual destruction of the liver. Some cases are caused by drugs, some by hepatitis B, some by post-transfusion hepatitis, but most have an unknown cause. Chronic active hepatitis may begin with a typical attack indistinguishable from acute viral hepatitis. In other cases, however, the illness begins gradually with a sense of fatigue, muscle or joint aches, nasal bleeding, easy bruising, or simply as the gradual discoloration of jaundice. The diagnosis is often delayed because of the mild, nondescript nature of the symptoms. A multiple-channel blood test that shows abnormal liver enzymes will be a tip-off. The diagnosis must be confirmed by a liver biopsy. The more severe cases are treated with cortisone-like drugs. The outcome is frequently favorable.

**Chronic persistent hepatitis** does not produce significant disease. Its major importance lies in possible confusion with chronic active hepatitis. The persistent form causes only a minor inflammation in the liver that does not progress. The liver biopsy is the only sure way of establishing whether a patient is suffering from chronic active hepatitis, which requires treatment, or from chronic persistent hepatitis, which requires no treatment.

## Alcohol and the Liver

Alcohol is one of the most common causes of severe liver injury in the United States. This does not mean that every social drinker suffers liver damage, for the liver can adjust well to moderate amounts of alcohol, and not all persons are equally susceptible. However, alcohol-related damage to the liver has become one of the nation's leading causes of death.

Changes in the liver brought about by alcohol occur in sequence. The earliest change is the fatty liver. The liver becomes large and pale, with a yellowish hue, and on autopsy the cells are found crammed with fat. This change produces no symptoms and few, if any, laboratory abnormalities. Continued heavy drinking can bring on inflammation of the liver tissue and death of some of the cells. This stage is called alcoholic hepatitis and does cause symptoms, primarily fever, jaundice, and abdominal pain. If the person stops drinking, the injury may be reversible. However, there is also a possibility that scarring of the liver (cirrhosis) may occur, regardless of whether drinking is stopped. Sometimes cirrhosis appears after a single episode of alcoholic hepatitis; sometimes it occurs after repeated episodes. The symptoms in this stage are fatigue, loss of energy, and swelling of the legs and abdomen.

## Cirrhosis of the Liver

Although heavy use of alcohol is the leading cause of cirrhosis in the United States, the term cirrhosis is not the same as alcoholic liver disease. There are many forms and many causes of cirrhosis. The disease is characterized by extensive scarring within the liver substance. In addition, there are areas of liver tissue where cells have regenerated to replace those damaged by the disease. The anatomical arrangement of these cells is abnormal. The new arrangements, called regenerative nodules, function normally but block the normal pattern of blood flow through the liver.

The methods of spread vary, but the organisms usually enter the body through the mouth. Infected persons who contaminate the food and food that comes from animal sources that carry these organisms are major sources of spread. (Most meats and poultry contain many of the bacteria that cause food poisoning, but the bacteria are killed if the foods are cooked adequately.) Contaminated water is a common source of organisms causing diarrhea, particularly in areas where the sewage disposal system is inadequate. Sometimes animals die and spread bacteria into the water. Homosexual activity has also been implicated in spreading the unwanted bacteria from person to person. Epidemics of amebiasis, giardiasis, infectious hepatitis, bacillary dysentery, and typhoid fever have all been traced to contamination of food or water spread from individual to individual.

### Typhoid Fever

Typhoid fever is a particularly severe form of salmonella infection caused by the strain known as *Salmonella typhi*. Symptoms include sustained high fever often lasting for several weeks, severe headache, constipation, a feeling of severe illness, and sometimes cough. Small red spots may appear on the abdomen. The major complications are intestinal hemorrhages. The intestines may be perforated by the bacteria, a serious problem indeed.

In contrast to other *Salmonella* strains that are widely found in domestic and wild animals, the typhoid bacillus lives only in humans. Because the bacillus is cast off from the body through bowel movements, it can contaminate food and water unless sanitary precautions are taken. In the past typhoid fever occurred in widespread epidemics because drinking water systems were contaminated by sewage. Today the water supplies are safe in most developed countries. When an outbreak of typhoid fever occurs, it is usually because a typhoid carrier (a person who is carrying the typhoid bacilli but shows no obvious symptoms) unknowingly contaminates food before it is eaten by others.

Persons who have recently recovered from typhoid fever sometimes continue to shed germs in the stool for weeks, months, or even for a lifetime. Many are not aware that they are typhoid carriers, although they are aware that they had the disease. Such carriers should observe special sanitary precautions. After

### Precautions to Prevent Gastroenteritis and Diarrhea

- Infected persons should not handle food others will eat.
- Wash hands after using the toilet and before preparation of food.
- Prepared food should be refrigerated if not eaten promptly.
- Meat and meat products should be cooked. Avoid rare and raw foods.
- Never eat pork and poultry when they are pink (not thoroughly cooked).
- A frozen turkey or large fowl should be completely thawed before being stuffed and roasted. If the interior of the turkey is not completely thawed, it may not cook thoroughly. Bacteria will multiply and gastroenteritis will spread.

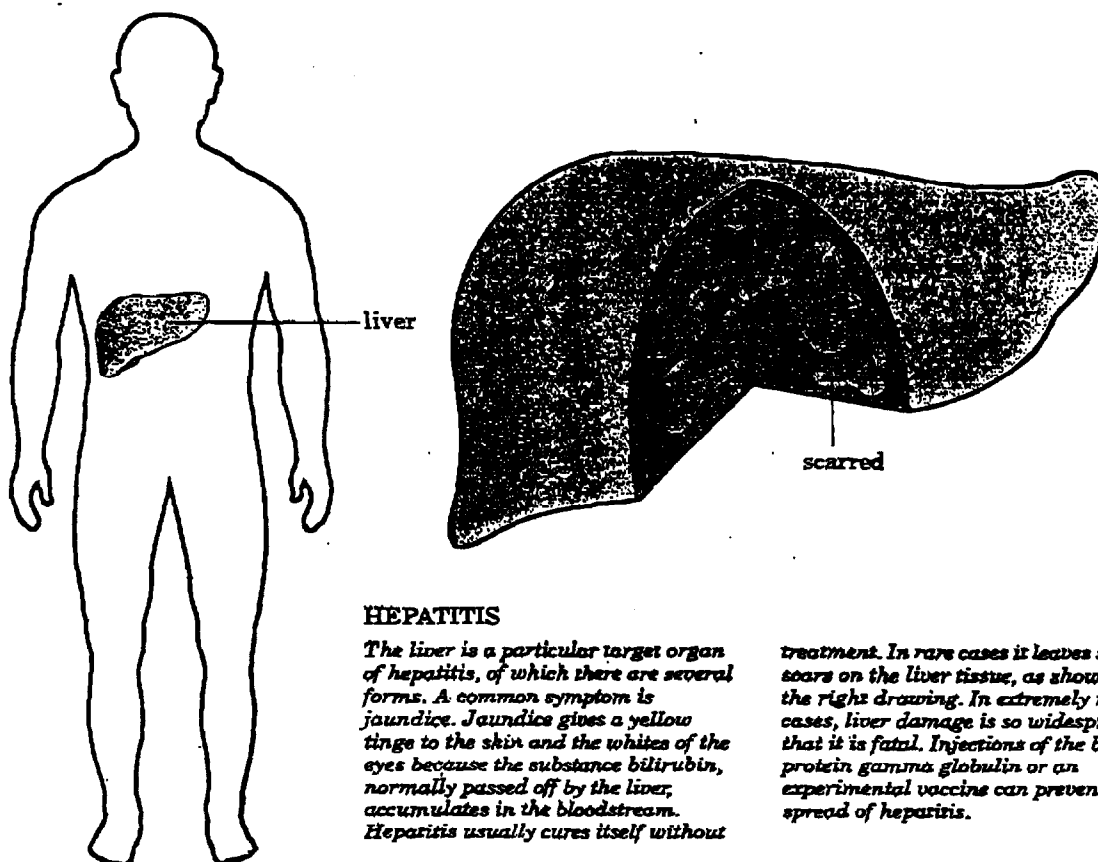
using a toilet, they should spray the seat and bowl with disinfectant. They should wash their hands carefully afterwards, and wash before handling foods. Known carriers should not be involved in the preparation or handling of food in restaurants.

Typhoid fever can be treated with antibacterial drugs, and a vaccine is available that lowers the risk of contracting the disease. A person traveling into areas where typhoid fever still occurs should be vaccinated as a precaution (see page 697). The vaccine is not effective if the person receives a large dose of typhoid bacilli in food or water, but it is effective against smaller doses. The vaccine itself often causes some swelling and pain of the arm and even a mild fever.

### Hepatitis

Research over the past few years has uncovered several different types of hepatitis. Two main forms have been definitely identified. A third form and perhaps additional ones are being recognized.

**Type A hepatitis.** "Infectious hepatitis" and "serum hepatitis" are the traditional names for the most common types. Infectious hepatitis is more precisely called type A hepatitis. The virus causing this disease usually spreads through bowel movements after being ingested much like the viruses of food poisoning. Within two to five weeks, susceptible persons who have acquired the virus will develop nausea, vomiting, loss of appetite, and loss of taste for cigarettes and many foods.



### HEPATITIS

*The liver is a particular target organ of hepatitis, of which there are several forms. A common symptom is jaundice. Jaundice gives a yellow tinge to the skin and the whites of the eyes because the substance bilirubin, normally passed off by the liver, accumulates in the bloodstream. Hepatitis usually cures itself without*

*treatment. In rare cases it leaves severe scars on the liver tissue, as shown in the right drawing. In extremely rare cases, liver damage is so widespread that it is fatal. Injections of the blood protein gamma globulin or an experimental vaccine can prevent the spread of hepatitis.*

They often develop intense feelings of fatigue and muscle and joint aches. The urine may become dark, the color of the stools more like clay, and jaundice may appear (yellowing of the skin and whites of the eyes). Sometimes there is mild fever. Children often have a milder illness and do not always develop jaundice, but they still spread the virus to others.

Type A hepatitis usually clears up within a week or two, but in some instances continues for several months. In a few relatively rare instances the disease progresses to cause severe scarring of the liver, known as cirrhosis. Convalescence needs vary. Some persons feel fit within a few days or weeks, while others require months to return to normal. Occasionally, those who have resumed full activity suffer a relapse. The illness sometimes reappears, although generally in a milder form. A rare complication of type A hepatitis is infection so severe that the liver is destroyed, causing death in less than a week.

There is no specific treatment for type A hepatitis except bed rest. Precautions similar to those for gastroenteritis and typhoid fever should be taken to avoid spreading the virus to others. Individuals with type A hepatitis are not likely to spread the disease to other members of their family or to those with whom they are in close contact. Nevertheless it is a good idea to have ill persons use separate dishes and to wash them separately in a dishwasher or in strong detergents to minimize any possibility of spread.

Type B hepatitis is often more severe than type A. The virus is most commonly spread from the blood of an infected individual to others, but it can be ingested in food or water, too. Type B hepatitis is a particular problem among homosexuals because it is spread by certain forms of sexual contact. Type B hepatitis usually takes longer to incubate. The disease often does not appear for six months after someone has acquired the virus. It is often slower to heal than type A, and is more likely to leave the person with scarring of the liver. A few persons retain type B virus in the

to the urethra, the conduit from the bladder

## INFECTIOUS DISEASES

blood for many years. Like type A, there is no specific treatment except bed rest.

Both types of hepatitis are accompanied by virus in feces, saliva, tears, and blood. In both instances unsanitary conditions can aggravate spread of the disease. Untreated sewage often contains hepatitis virus because so many people pass the virus out in their feces. Hepatitis outbreaks have been traced to shellfish taken from waters contaminated by untreated sewage. Narcotics addicts who share needles without sterilizing them are also particularly likely to pass type B virus to one another. Carriers of type B hepatitis virus should never be permitted to give blood for transfusions. Carriers can be detected by a simple blood test.

Protection against the spread of hepatitis, particularly type A hepatitis, can be achieved by injections of human gamma globulin, a blood protein that carries antibodies against the virus and that can neutralize the infectious particles before they spread. In recent years scientists have developed a vaccine against type B hepatitis. Still in the experimental stage, the vaccine appears to be effective, although it takes two or three months after injection for the effect to be fully realized.

Blood tests can distinguish type A and type B hepatitis. The use of the tests has disclosed another form of hepatitis that is transmitted by blood products and is neither A nor B. It has been termed "non-A/non-B hepatitis," but in fact it may represent a group of infections. Research in non-A/non-B hepatitis has been helped by the discovery that this form of hepatitis can be produced in chimpanzees.

Many viruses other than the ones usually associated with hepatitis can spread to the liver and produce jaundice. Infectious mononucleosis (see page 690) is often associated with jaundice, as is a disease called cytomegalic inclusion disease. In fact, any severe infection can produce some degree of jaundice.

## URINARY INFECTIONS

Urinary infections are among the most common of all infections, occurring in 10 to 20 percent of women and in about one-half of one percent of men at some time in their lives. The infections are often present without causing symptoms, so specific tests have been developed to detect them.

Infections of the urinary system are usually caused by the common bacteria found in the intestinal tract. These bacteria find their way to the urethra, the conduit from the bladder

that leads outside the body. The infection is quite short, open to treatment. Bacteria ascend the urethra and multiply in the bladder and multiply in the urethra inhibit the greater length probably explains why it is much less common.

No one knows why urinary tract infections do not. Sometimes by the use of catheters but this accounts for infections in women. account for the infections in men.

The symptoms of urinary tract infections consist of a frequent need to urinate, often at night with discomfort and pain in the lower back and often infected, too. When symptoms are the same as the above there also can be febrile area.

Infections of the urinary tract are about one percent of all infections. by about one percent of women. that at any given time about one percent of women have a urinary infection. Many urinary tract infections are only to re-infect the number of women who have had urinary infections. the number infected. figures suggest that the infection is through which women get urinary infections. no one knows why. infections in which the bladder is involved.

Although bladder infections can follow instrumental procedures, result from enlargement of the prostate. The prostate is located below the bladder through which the urine is discharged. Enlargement of the prostate is common in men over 50 years of age. emptying of the bladder is difficult and if not all of the bacteria will be removed and can spread to the kidneys. difficult to eradicate if there is obstruction or if the infection interferes with the